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ON THE STUDY OF HEMOSPERMIA

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CHAPTER 1. INTRODUCTION

Hemospermia appears as a presenting symptom or as one of the concomitant symptoms in various diseases of the seminal tract. Recently, a fairly large number of case reports have become available on this condition. Many of them, however, regard asymptomatic bloody semen as a unique indication, and not a few points of this condition still remain ignored.

On the other hand, a variety of opinions have long been discussed as to the cause of hemospermia, but there are still many problems left unsolved. Consequently, no satisfactory report has yet been made on the treatment of this condition. Of late, seminal vesiculography and other urological examinations have indeed made a great improvement, but still we often encounter hemospermia of unknown etiology. It is also experienced that some cases are compelled to undergo the surgical removal of the seminal vesicles and vas deferens owing to prolonged or severe hemospermia.

In recent years the relationship of hemospermia to male infertility and further to allergy of the seminal vesicles has been acquiring a great importance. In this sense, etiological solution of this condition has been considered as one of the most important problems.

Hereupon, in order to investigate the

relations between hemospermia and male infertility, we made clinico-statistical observations on the patients with hemospermia who visited Department of Urology, Osaka Medical College. In connection with this observation, experimental studies were made on the effect of various sera, the seminal plasma of patients with seminal vesiculitis or prostatitis upon the motility of sperm. In addition, based on the study of our cases of unknown cause, the presence of seminal vesicle allergy was conjectured as one of the causes of this condition, and various animal experiments concerning the problems were carried out obtaining noticeable results.

CHAPTER 2. BIBLIOGRAPHIC
CONSIDERATION

A large number of reports have contributed to the problem on hemospermia, but there are still many uncertain aspects. Hereupon, it would be necessary to discuss the reports of other investigators on hemospermia and to give some considerations to the etiology of this condition.

Originally, hemospermia is divided into two types: genuine hemospermia and pseudo-hemospermia. Guelliot (1883)¹⁾ stated that bleeding came from the spermatogenetic organs in the former and from the urethra in the latter, and that the former genuine one might originate

in the seminal vesicles. In opposition to this theory of seminal vesicle bleeding, there is a theory of prostatic bleeding advocated by Keersmecker (1899)²⁰. Still at present, however, many investigators consider the seminal vesicles to be a hemorrhagic origin.

According to Voelcker (1912)³¹, hemospermia was described for the first time by Peterson in 1670 as "ejaculatory fluid as black as ink." Afterwards, Neuman (1837)⁴¹ reported the bleeding from the seminal vesicles as "spermatocystidorrhagie" in his work titled as "Spermatocystitis." Rapin (1859)⁶¹ stated that the discharge of bloody semen was one of the important symptoms in secondary spermatocystitis. Guelliot¹¹ considered that hemorrhage in hemospermia was mostly attributable to the seminal vesicle. As the ground for it, he obtained a blood-containing fluid from the seminal vesicles on autopsy of an old man. Such hemorrhage was, in his opinion, a result of stagnation of the fluid, and he further stated that it might be caused by chronic spermatocystitis. From the anatomical point of view, Paul Cohn (1907)⁶¹ considered that the seminal vesicles consisting of a single layer of mucosal epithelia, rich in blood vessels and capillaries might be responsible for the bleeding, and he examined into this organ to search for the cause of this condition. Of late, Herbst & Merrick (1944)⁷¹, who carried out catheterization of the seminal vesicle under the diagnosis of spermatocystitis, recognized bloody semen as one of its symptoms. Besides, Magid & Hejtmanick (1957)⁸¹ performed catheterization of the ejaculatory duct in two patients with hemospermia, and

accidentally discovered a picture of communication between the pelvic venous system and the seminal vesicles on the obtained retrograde seminal vesiculograms. They regarded this communication as a cause of hemospermia. Although the etiological mechanism was uncertain, they further stated as follows. Probably because of a thin layer of the seminal vesicles and coexistent chronic spermatocystitis and prostatitis seen in both cases, erosion and ulcer were formed on the wall of the seminal vesicles, which might have produced a communication passage between the seminal vesicles and the veins. Huggins & McDonald (1945)⁹¹ reported 20 cases of hemospermia in their paper discussing chronic hemospermia.

They reported that gross examination of the fractionated ejaculate collected in three glasses showed little or no blood in the first glass and progressively greater amounts of blood in the second and third glasses.

The concentration of glucose, a component arising from the seminal vesicle, was found to be greatest in the third glass whereas the prostatic portion, fibrinolysin and acid phosphatase were greatest in the first glass, and it was found that the hemorrhage arose from the vesicular apparatus. In our country, Nakao (1936)¹⁰¹ carried out seminal vesiculography in 4 patients with hemospermia, and observed abnormal findings on the obtained radiograms in all of the cases; spermatocystitis was proved to be responsible for 3 cases and vascular sclerosis 1 case. He concluded that bloody semen was a result of hemorrhage from the seminal vesicles in almost

all cases of genuine hemospermia, but he did not express any objection to the opinion insisting hemorrhage from the prostate and other organs.

On the other hand, contrary to the theory of bleeding from the seminal vesicles, Keersmecker⁸⁾ published his paper concerning "Håmospermie". In this paper, he advocated a theory of prostatic bleeding, considering that bleeding had resulted from hemorrhagic prostatitis in all his 9 cases of hemospermia. In other words, he said that he could not obtain any abnormal findings in the seminal vesicles on rectal palpation and that bloody semen was demonstrated in the discharge obtained by prostatic massage. B. Goldberg (1903)¹¹⁾ likewise, asserted that hemospermia was caused by the lesion of the prostate. Zuckerkandle¹²⁾ concluded that hemospermia was not caused by spermatocystitis but by chronic prostatitis. Hamada (1936)¹³⁾ of our country, made a report on a 48-year-old patient with hemospermia, and reported that the prostatic fluid obtained by pressure grossly attired a color of blood, so he presumed it might be the result of the bleeding from the prostate. It would be, however, thoughtless to regard the discharge obtained by prostatic massage immediately as the secretion of the prostate as Keersmecker and Hamada did. We should consider the possibility of mixing of the content of the seminal vesicles in the obtained discharge to some extent. In addition, it would be necessary to make further close examinations before we made a decision that the seminal vesicle was healthy. From such points of view, there are still some suspicious points in the

theory of prostatic hemorrhage.

Momose et al. (1961)¹⁴⁾ carried out seminal vesiculography in 13 cases with hemospermia, and considered the seminal vesicles as a principal hemorrhagic lesion on the basis of the findings obtained by various examinations and of histopathological findings on resected seminal vesicles.

Considering from the bibliographical studies described above, we are not able to deny a role of the prostate in the cause of hemospermia, but it would be rather plausible to regard the seminal vesicle as a main hemorrhagic organ.

In the next place, we review historically the investigations about the disease causing hemospermia. First, as organic diseases there are specific inflammations of the seminal tract. Among them, genital tuberculosis was examined as to its relation to this condition. Eisendrath & Rolnick (1928)¹⁵⁾ reported that hemospermia was often associated with tuberculosis of the seminal vesicles and prostate. Hugh Youngs (1926)¹⁶⁾ expressed his opinion that hemospermia originated from tuberculosis of the prostate. In addition, Voelcker³⁾ said he had often observed the mixture of blood in the semen discharged from the patient with tuberculosis of the prostate. Walthard (1927)¹⁷⁾ stated that hemospermia appeared in the case of tuberculosis of the seminal vesicles. Kondo (1959)¹⁸⁾ reported that the semen turned out brown due to blood in tuberculosis of the seminal vesicles. Parker (1942)¹⁹⁾ regarded tuberculosis of the seminal vesicles as one of the important causes of hemospermia. In addition, Inada (1949)²⁰⁾ reported that hemospermia was

an incipient symptom of prostatic tuberculosis, and that it could be observed when the seminal vesicles were not affected. Hatono (1960)²¹⁾ carried out an occult blood test on the prostatic secretions obtained by prostatic massage; the finding was negative in 46 cases which had no abnormality in the prostate, and positive in 15 out of 25 cases of tuberculosis of the prostate. He reported clinical significance of hemospermia as a supplementary diagnosis of prostatic tuberculosis. His paper similarly insisted that bleeding came from tuberculosis, but as the above criticism on Keersmecker's prostatic hemorrhagic theory, it is nearly impossible with such a report to give up completely the theory of seminal vesicle hemorrhage, and moreover hemospermia can be hardly defined as a bleeding from the prostate.

As for genital syphilis, Paul Cohn⁶⁾ reported a case of hemospermia caused by syphilis of the seminal vesicles in his paper "Über Hämospermie; ein Fall von Lues haemorrhagica der Samenblasen" in 1907. In our country, Arakawa et al. (1944)²²⁾ also reported a case of hemospermia caused by syphilis of the seminal vesicles. In their report of a 51-year-old patient, the seminal vesicles were felt hard and any anti-inflammatory treatment was not effective, but antisiphilitic therapy eradicated the symptom of hemospermia.

It is also reported that hemospermia appearing as a symptom of nonspecific chronic seminal vesiculo-prostatitis has peculiarity. Asahi (1926)²³⁾ reported a case of hemospermia in the report concerning spermatocystitis. Ochiai (1959)²⁴⁾ who considered that hemospermia was a

peculiar symptom of chronic seminal vesiculo-prostatitis, carried out seminal vesiculectomy in a case with hemospermia which had been diagnosed as chronic spermatocystitis in order to make a histopathological examination, but he could not demonstrate any significant changes. He emphasized the necessity of studying the case on clinical symptoms, seminal vesiculograms, the finding of operation and further on histological findings.

As regards the morphological anomalies of the seminal vesicles and the vas Kusunoki (1947)²⁵⁾ reported a case of diverticulum of the seminal vesicle with a chief complaint of hemospermia. Since then, many case reports have been made in succession, for instance, by Stewart & Nicoll (1949)²⁶⁾, Nakao (1952)²⁷⁾, Ishigami (1953)²⁸⁾, Nakamura et al. (1955)²⁹⁾, Nakajima & Yanase (1958)³⁰⁾, Shimoe (1959)³¹⁾, Kanazawa & Fukuda (1959)³²⁾, Ishigami (1960)³³⁾, Nagata & Mizumoto (1961)³⁴⁾, Ishikawa & Jono (1962)³⁵⁾ and Moriwaki & Yuen (1962)³⁶⁾. In those who had operation hemospermia disappeared. When added my 9 cases, nearly 40 cases reported as cyst or diverticulum are to be the cases which showed hemospermia. In accordance with the recent advances in the diagnostic technique, the relation between homospermia and abnormal dilatation of the seminal vesicles and seminal ducts will be further elucidated. Among the anatomical anomalies is the direct communication between the seminal vesicles lumen and the venous system. Magid & Hejtmanick⁸⁾ accidentally observed the communication between the seminal vesicles and the pelvic veins on the

seminal vesiculograms obtained in 2 cases with hemospermia, and concluded that the direct communication was the cause of hemospermia. A similar case was also observed by author. As to the cause of the communication, Magid & Hejtmanick⁸⁹ postulated the formation of ulcer in the wall of the seminal vesicle due to inflammation.

In the next place, there is hemospermia resulting from seminal vesiculolithiasis, calcification of the seminal vesicles and the vas deferens.

There are but a few reports on it. Namiki (1960)⁸⁷⁾ reported a case of seminal vesicle calcification with a chief complaint of hemospermia. On a plain film (KUB) he recognized stones and calcification which seemed to be due to tuberculosis of the seminal vesicle and carried out seminal vesiculectomy. As the result, he could remove a large number of stones and demonstrate histologically the calcification in the epithelium of the seminal vesicles. Momose et al.¹⁴⁾ reported that calcification in tuberculosis of the seminal vesicles was diagnosed in 1 out of 13 patients with hemospermia. Voelcker³⁾ stated that hemospermia should be expected to occur by the effect of the stones in the seminal vesicles. As to the cause of such stones, Kawai (1960)⁸⁸⁾ stated that the retention of the seminal vesicular fluid as a result of inflammatory occlusion of the seminal ducts and the subsequently occurring inflammation might cause the precipitation of minerals with the epithelium of the seminal vesicles, inflammatory cells or sperms as nidi of stone formation. Momose et al. also considered the stagna-

tion and concentration of the seminal vesicular fluid resulting from the dilation of the glandular cavity as a cause of stone formation.

Besides, there are reports on hemospermia due to tumor, trauma and others. Hugh Young¹⁶⁾ insisted the possibility of occurrence of hemospermia in prostatic tumor and prostatic hypertrophy.

As to hemospermia caused by trauma, Omori and Kamimura reported their cases respectively. In all their cases, secondary disturbance due to trauma not injury itself, led to the discharge of bloody semen. Omori (1952)³⁹⁾ reported a case of intraepididymal sperm invasion and inflammatory granuloma in association with bloody semen, in which the wall of the epididymis was damaged by trauma and sperm invaded out of the epididymal tubules causing prolonged hemospermia for as long as 13 years. Kamimura (1955)⁴⁰⁾ reported a case of bloody semen seen after the contusion in the perineal region and he could observe a localized bleeding lesion in the epididymis and overflow of the sperm on operation. Parker¹⁹⁾ reported a case of hemospermia due to testicular trauma.

Nakano (1950)⁴¹⁾ described on 13 cases of hemospermia and stated that many of those cases had once suffered from gonorrhoea, particularly from epididymitis in their past. Ashker and Issa (1935)⁴²⁾ reported 7 cases with hemospermia due to bilharziasis.

In addition to the hemospermia that are considered to be caused by organic diseases, there are some reports of the cases which are attributable to functional disturbances. Tomikawa (1939)⁴³⁾ collected the content of the seminal vesicles

in 51 cases by catheterization of the ejaculatory ducts, and demonstrated erythrocytes in 9 cases. Of his 9 cases, 2 cases were reported to be the patients of sexual neurasthenia. Nakano⁴¹⁾ reported a case of hemospermia developed in the patients with sexual neurasthenia.

As for the cases which seem to be due to sexual excess, Guelliot¹⁾ reported a 23-year-old doctor who had 11 times of coitus in one night finally ejaculating only a few drops of blood. Parker¹⁹⁾ insisted that excess in masturbation and sexual intercourse might be the cause of hemospermia.

Nelken⁴⁴⁾ and Shorpsire⁴⁵⁾ reported their works under the title "essential hemospermia" in 1910 and 1912, respectively.

In 1957, Ishigami⁴⁶⁾ also reported a case of hemospermia without organic changes and presumed the existence of "essential seminal vesicle bleeding" from the histological finding obtained by seminal vesiculectomy on the case. Allergic changes of the blood vessels were observed in the seminal vesicles on autopsy of the patient diagnosed as allergic prostatic granuloma in one case of Melicow (1951)⁴⁷⁾. In 1962, Momose et al¹⁴⁾ recognized a change similar to allergic reaction in the tissue extracted from the seminal vesicles, and suspected of seminal vesicle allergy as a cause of hemospermia. They advocated to tentatively designate such hemospermia of functional origin as "essential hemospermia."

Some considerations on the hitherto reports and findings concerning hemospermia were above mentioned. In short, hemospermia is a sign observed as a

main or one of the concomitant symptoms of the diseases in the seminal passage. The hemorrhagic lesion chiefly lies in the seminal vesicles, but it can not be denied the bloody semen is originated from the prostate. In addition, it would be very convenient to discuss the cause of hemospermia from the organic and functional disturbances of the seminal tract.

CHAPTER 3. CLINICAL DATA

In the present paper, the author report the statistical observations on the patients who came to the urological clinic of Osaka Medical College during the past 4 years complaining of hemospermia as a main or one of the concomitant symptoms and on the patients who came to hospital with male infertility or other diseases and were thereafter found to have hemospermia as the result of examinations; and the results of various examinations in every cases were also referred.

ARTICLE 1. STATISTICAL OBSERVATIONS

Total number of the ambulant patients in the clinic amounts to 3,898 in the past 4 years. Of them, there were 33 cases of hemospermia (0.84%). The outline of the clinical process and the results of various laboratory examinations in all these cases are shown in Table 1, 2 and 3. As for the age distribution, the largest number of cases were seen in the young to middle aged group with the average age of 34. When classified by profession, company employee or the person who engage in office works or the like are a little larger than the other profession in number. As for the chief

Table 1. Clinical features of hemospermia (33 cases).

No.	Patient	Age	Chief complaint	Duration of hemospermia	Marriage	Number of children	Frequency of intercourse/Wk	Associated symptoms	Past history
1	Y. H.	38	Hemospermia	3Wks.	Yes	2	2	None	Pulmonary tuberculosis
2	Y. M.	24	Hemospermia	1Wk.	No	0	?	None	None
3	K. O.	33	Hemospermia	2Wks.	Yes	1	1	None	None
4	S. O.	35	Sterility	3Mos.	Yes	0	2	None	Pulmonary tuberculosis
5	T. M.	31	Hemospermia	2Wks.	Yes	1	2-3	None	None
6	T. A.	26	Hemospermia	3Mos.	No	0	?	None	Gonorrhea
7	S. K.	23	Hemospermia	2Wks.	No	0	?	Perineal discomfort	Gonorrhea
8	K. H.	31	Hemospermia	3Mos.	Yes	0	2	None	None
9	Y. T.	40	Hemospermia	2Mos.	Yes	2	1	None	Pulmonary tuberculosis
10	S. S.	27	Hemospermia	8Mos.	Yes	1	2-3	None	None
11	J. I.	36	Hemospermia	16Yrs.	Yes	2	2	Perineal discomfort. Ejaculatory pain	Gonorrhea
12	Y. O.	36	Hemospermia	1Wk.	Yes	3	2	None	None
13	T. K.	34	Sterility	1Wk.	Yes	0	3-4	None	None
14	T. K.	54	Hemospermia	1½Mos.	Yes	2	1	None	Pulmonary tuberculosis
15	T. S.	53	Hemospermia	3Ds.	Yes	4	1	None	Gonorrhea
16	K. T.	26	Hemospermia	2Wks.	No	0	?	None	None
17	Y. U.	33	Hemospermia	2Wks.	Yes	2	1	None	Right-epididymitis
18	S. Y.	26	Hemospermia	2Mos.	Yes	0	3	None	Pulmonary tuberculosis
19	K. O.	34	Hemospermia	1Mo.	Yes	2	2-3	None	None
20	S. T.	24	Hemospermia	3Wks.	Yes	1	2-3	Perineal discomfort	Gonorrhea
21	M. K.	20	Hemospermia	2Wks.	No	0	?	None	None
22	Y. T.	24	Hemospermia	1Mo.	No	0	?	None	None
23	S. N.	36	Hemospermia	1Mo.	Yes	2	2	None	Pulmonary tuberculosis
24	Y. H.	29	Sterility	1Mo.	Yes	0	2-3	Perineal discomfort	None
25	S. T.	24	Hemospermia	3Mos.	No	0	?	Perineal discomfort	None
26	Y. K.	29	Sterility	3Wks.	Yes	0	2-3	None	None
27	S. K.	28	Hemospermia	3Wks.	No	0	?	Lumbago	None
28	Y. T.	42	Hemospermia	2Mos.	Yes	2	1	Lumbago	None
29	Y. K.	65	Hemospermia	35Yrs.	Yes	3	1	None	Gonorrhea
30	T. Y.	24	Pain of testicle	1D.	Yes	1	2	Pain of 1-testicle	Contusion of scrotum. Parotitis
31	H. Y.	49	Hemospermia	1Mo.	Yes	3	1	None	Pulmonary tuberculosis
32	M. Y.	43	Hemospermia	1Mo.	Yes	2	1	None	None
33	M. M.	45	Hemospermia	1Yr.	Yes	1	0	None	Pulmonary tuberculosis

complaints, homospermia was reported in 27 cases (81.8%), the largest number of case; infertility in 5 cases (15.1%) and testicular pain in 1 case (3.0%). The discovery of this condition was made in almost all the cases by macroscopic observation of the semen obtained by means of intercourse with condom or masturbation. In the cases with the chief complaint of infertility, homospermia was first discovered or pointed out as the result of the examination at hospital. As the coexisting symptoms of homospermia, there have been lower quadrant abdominal pain, a sense of discomfort in the perineal region and in the urethra and pain on ejaculation.

In addition, some cases are completely free from concomitant symptoms. In my experience, a sense of discomfort in the perineal region was complained of in 5 cases (15.1%), dull pain in the lumbar region in 2 cases (6.0%), and testicular pain in 1 case. There were no concomitant symptoms at all in the remaining 25 cases. Particularly in the case of abnormal dilatation of the seminal vesicles which has been discussed as one of the causes of homospermia, a relatively large cyst is reported to cause oppression symptoms, but in cases, however, such symptom have not encountered. As for past history, pulmonary tuberculosis was noted in 8 cases (24.2%), gonorrhea in 5 cases (15.1%) and simple orchitis in 1 case (3.0%). Tuberculin reaction was performed in 26 cases and turned out to be positive in all the cases. Serologic study for syphilis proved to be negative in all the cases. Both bleeding time and coagulation time were normal in all the cases. When classified as to

marital state, married patients were overwhelmingly larger in number than the unmarried in the ratio of 25 to 8. As for the sexual life, one to four sexual intercourse a week was common. It is of great interest that nearly half of the patients complained of decreased sexual activity since they became aware of this condition.

Momose's report saying that a decrease in sexual activity was observed in all his cases of homospermia. It is, however, uncertain at present whether this is the result of organic disturbance or of ensued psychoneurosis.

Five out of 25 married patients complained of infertility, but the other cases were all the fathers of healthy children, the number of which being 1 to 4. However, it was before the onset of this condition that they became father; and since the occurrence of homospermia their wives have not become pregnant. No history of diabetes was noticed in any case, and sugar in urine was negative in all the cases.

In the examination of the semen, the color of the semen was grossly seen to be pink to chocolate brown. The volume of semen was 1 to 5cc. There was no case with too little or too much semen. Sperm count was 0 to $113 \times 10^6/\text{cc}$. Azoospermia, oligozoospermia or necrozoospermia were evidently shown in 12 cases (36.3%). Therefore, it is evident that many cases are considered to be in a state of infertility. Moreover, the motility of the sperm was observed to be a little lowered even in the case with normal sperm count, needless to say of oligozoospermia. Erythrocytes were

Table 2. Semen examination of hemospermia.

No.	Patient	Color	Microscopic examination					Bacteriologic examination (Culture)	
			Volume (cc)	Sperm count ($\times 10^6$ /cc)	Sperm motility (%)	Erythrocyte	Leucocyte	Tubercle bacillus	Bacteria
1	Y. H.	Pink	2.0	23	9.3	++	+	Negative	Pneumococcus
2	Y. M.	Brighterred	2.5	60	56.0	++	+	Negative	Pathogenic staphylococci
3	K. O.	Pink	3.0	53	63.3	++	—	Negative	Negative
4	S. O.	Pink	3.5	0	0	+	+	Negative	Pathogenic staphylococci
5	T. M.	Brighterred	4.3	23	39.4	++	++	Negative	Pathogenic staphylococci
6	T. A.	Pink	1.8	8	54.8	++	++	Negative	Pathogenic staphylococci
7	S. K.	Pink	1.3	21	76.0	++	—	Negative	Enterococcus
8	K. H.	Pink	2.2	93	76.9	++	—	Negative	Pathogenic staphylococci
9	Y. T.	Pink Clottedblood	2.8	78	75.1	+	+	Negative	Pathogenic staphylococci
10	S. S.	Brighterred	2.5	86	77.0	+++	+	Negative	Pathogenic staphylococci
11	J. I.	Brighterred	2.0	36	70.0	+++	+	Negative	Pathogenic staphylococci
12	Y. O.	Chocolatebrown	1.0	50	0	+++	+	Negative	Pathogenic staphylococci
13	T. K.	Pink	2.0	0	0	+	+	Negative	Pathogenic staphylococci
14	T. K.	Chocolatebrown	1.5	2	0	+++	+	Negative	Negative
15	T. S.	Pink	1.8	70	60.0	++	—	Negative	Negative
16	K. T.	Pink	2.5	80	80.0	++	+	Negative	Negative
17	Y. U.	Pink	2.0	36	60.0	++	—	Negative	Negative
18	S. Y.	Pink	2.5	50	0	++	—	Negative	Negative
19	K. O.	Chocolatebrown	3.0	65	50.0	++	+	Negative	Pathogenic staphylococci
20	S. T.	Pink Clottedblood	2.0	80	56.0	+	+	Negative	Pathogenic staphylococci
21	M. K.	Pink Clottedblood	2.3	113	48.0	+	+	Negative	Pathogenic staphylococci
22	Y. T.	Chocolatebrown	2.5	80	60.0	++	+	Negative	Pathogenic staphylococci
23	S. N.	Pink	3.0	83	56.0	++	+	Negative	Pathogenic staphylococci
24	Y. H.	Chocolatebrown	5.0	0	0	++	+	Negative	Negative
25	S. T.	Chocolatebrown	5.0	60	30.0	+++	+	Negative	Streptococci
26	Y. K.	Pink	2.4	0	0	+	+	Negative	
27	S. K.	Pink	3.0	95	50.0	+	+	Negative	Negative
28	Y. T.	Pink	3.5	50	30.0	+	+	Negative	
29	Y. K.	Chocolatebrown	2.0	10	0	++	+	Negative	
30	T. Y.	Pink	4.0	3	0	+	+	Negative	Pathogenic staphylococci
31	H. Y.	Chocolatebrown	3.0	30	0	++	+	Negative	Pathogenic staphylococci
32	M. Y.	Brighterred	3.0	85	45.0	+	+	Negative	Pathogenic staphylococci
33	M. M.	Pink	2.0	0	0	+	+	Negative	Pathogenic staphylococci

* +: 1-10/F. ++: 10-20/F. +++ over 20/F.

Table 3. Physical examination of hemospermia.

No.	Patient	Seminal vesiculogram	Rectal examination		Palpation
			Seminal vesicle	Prostate	Epididymis & testicle
1	Y. H.	Normal	Tenderness	Tenderness	Normal
2	Y. M.	Normal	Not palpable	Normal	Normal
3	K. O.	Normal	Not palpable	Normal	Normal
4	S. O.	Normal	Not palpable	Normal	Normal
5	T. M.	Normal	Not palpable	Normal	Normal
6	T. A.	Normal	Not palpable	Normal	Normal
7	S. K.	Normal	Not palpable	Enlargement	Normal
8	K. H.	Normal	Not palpable	Normal	Normal
9	Y. T.	Normal	Not palpable	Normal	Normal
10	S. S.	Normal	Not palpable	Normal	Normal
11	I. I.	Dilatation with stones of left ejaculatory duct	Cystic	Tenderness	Swelling of l-epididymis
12	Y. O.	Normal	Not palpable	Normal	Normal
13	T. K.	Normal	Not palpable	Normal	Normal
14	T. K.	Normal	Not palpable	Normal	Normal
15	T. S.	Normal	Not palpable	Normal	Normal
16	K. T.	Normal	Not palpable	Normal	Normal
17	Y. U.	Normal	Not palpable	Tenderness	Normal
18	S. Y.	Dilatation of right seminal vesicle	Tenderness	Normal	Normal
19	K. O.	Seminal vesicle	Not palpable	Normal	Normal
20	S. T.	Dilatation of right ejaculatory duct	Tenderness	Normal	Normal
21	M. K.	Normal	Not palpable	Tenderness	Normal
22	Y. T.	Normal	Not palpable	Normal	Normal
23	S. N.	Normal	Not palpable	Normal	Normal
24	Y. H.	Dilatation of seminal vesicle and ejaculatory duct	Cystic	Normal	Normal
25	S. T.	Dilatation of Müller's duct	Tenderness	Normal	Normal
26	Y. K.	Normal	Not palpable	Normal	Normal
27	S. K.	Dilatation of left seminal vesicle	Swelling	Normal	Normal
28	S. K.	Dilatation of both seminal vesicle	Swelling	Normal	Normal
29	Y. K.	Dilatation with stones of ejaculatory duct	Tenderness	Normal	Normal
30	T. Y.	Dilatation of seminal vesicle Seminal vesicle-venous communication on right side	Cystic Tenderness	Normal	Swelling of l-testicle
31	H. Y.	Normal	Not palpable	Normal	Normal
32	M. Y.	Normal	Not palpable	Normal	Normal
33	M. M.	Seminal vesiculitis	Tenderness	Normal	Normal

observed in all the cases. Pus cells were demonstrated in 27 cases (81.8%). In the culture of the semen, tubercle bacillus was negative in all the cases. As for the common bacteria, pathogenic staphylococcus was demonstrated in 19 cases, streptococcus in 1 case, pneumococcus in 1 case, and enterococcus in 1 case.

In seminal vesiculography, 22 cases (66.6%) were normal occupying the majority of the cases. Pathological dilatation was seen in 9 cases (27.2%), of which 2 cases had stones, 1 had seminal vesicle venous communication and 2 showed findings of inflammation. There was no case showing tuberculous changes particularly. As for the findings of the genital organs other than the seminal vesicles, tenderness and swelling of the prostate was demonstrated by digital palpation in 5 cases which, however, showed no evident inflammation.

It was only in one case that evident finding of inflammation in the testicle and epididymis was observed. The posterior urethra and the verumontanum showed hyperemia in two cases but no evident proof of urethral bleeding was obtained for differential diagnosis of hemospermia.

CASE REPORTS (REPRESENTATIVE CASES)

Case 11. J. I., 34-year-old male, president of a company.

Chief complaint : Hemospermia.

Past history : Gonorrhea.

Family history : Non-contributory. Two children.

Present illness : About in 1955 he noticed that the ejaculate was bloody, but he left it untreated. Hemospermia

became gradually severer, and finally he became to suffer from the passage of stones several times together with pain on ejaculations. In addition, he felt diminished sexual desire of late.

Status presents : No significant findings were obtained in the general physical examination except for slight swelling of the left epididymis. The left vas deferens could not be palpated. He had repeatedly been administered antibiotic solution through the vas at another hospital for the therapeutic purpose. It might be possible that the vas had been cut down by these procedures. Rectal examination revealed the prostate to be slightly enlarged, of normal consistency and smooth surface. Above and to the left of the midline, a fairly large, soft, fluctuant mass could be felt. It was slightly movable and was very tender.

Laboratory findings : The blood pressure was 120/80 mmHg in the lying position and 100/68 mmHg in the erect position. Hematologic examination showed 4,490,000 RBC, 112% Hb (Sahli's method), color index 1.25, 195,000 thrombocyte, 46% Hct and 7,200 WBC composed of 52% neutrophils, 1% acidophils, 0% basophils, 43% lymphocytes and 4% monocytes. Bleeding time was 1 minute. Coagulation time was 15 minutes. Serologic tests for syphilis were negative. Tuberculin test was positive (double flare). Blood NPN was 31.6 mg/dl. Urinary nitrogen excretion was 16.6 mg/dl.

Urinalysis : Yellowish transparent in appearance, neutral, negative in protein and sugar, and normal in urobilinogen, red blood cell (+), white blood cell (-), epithelium (-), cast (-), mineral crystal (-) and bacteria (-). Urine sediment

after prostatic massage turned out to be red blood cell (++) , non-motile sperm (+) and white blood cell (+).

Examination of the semen (collected by masturbation) demonstrated that the semen was apparently dark brown and jellied, 2.0 cc in volume containing 36×10^6 /cc sperm, with 70% motility, red blood cell (+++) and pus cell (+). Culture of the semen showed negative in tubercle bacillus and positive in pathogenic staphylococcus.

The capacity of the bladder was demonstrated to be 250 cc with normal mucosa by cystoscopy. In the indigocarmin test, excretion started at 3 minutes on the right and at 5 minutes on the left. Examination of renal function demonstrated 65% P.S.P. excretion. The posterior urethroscopy revealed no noticeable pathological changes. Chest x-ray was normal and no stone shadow was found in KUB. Excretory pyelography disclosed no abnormality in the pelves and ureters. As above mentioned, the vas deferens on the left had been cut down at approximately 2 cm. from the epididymis. On the left side the plastic operation of the vas was performed and at the same time 5 cc of 70% Endografin was injected into the both vasa obtaining seminal vesiculogram which was normal on the right side but on the left side showed Graaf's phenomenon being negative.

An abnormal shadow of a thumb size was observed in the portion corresponding to the part of the ejaculatory duct, and at the same time several ston-like shadows were recognized (Fig. 2). Diagnosis: Pathological dilatation of the left

ejaculatory duct with stones. Operative findings: A cyst with the size of a thumb tip was observed at the site of the left ejaculatory duct in which brown liquid content was recognized. After extraction of 10 stones the dilated portion was removed off and plastic operation was carried out to preserve fertility of the patient (Fig. 3).

Histological findings: The mucosal epithelium fell into severe degeneration, losing its proper structure and led to hemorrhage and slight necrosis. On the other hand, thickening of the wall of the arterioles lying under the mucosal membrane was so serious that the arterioles ran tortuously and slight edema was recognized in the surrounding connective tissue (Fig. 4).

Examination of the semen carried out 1 month after the operation showed the sperm count of 40×10^6 /cc, sperm motility of 75% and no demonstration of red blood cells. In the 14th month after the operation, the patient's wife gave birth to a healthy boy. He has not complained of recurrence of hemospermia.

Case 19. K. O., 34-year-old male, company employee.

Chief complaint: Hemospermia.

Past history & Family history: Non-contributory. He has two children.

Presens illness: He first noted hemospermia in September, 1961 and was hospitalized for 1 month. On admission no findings suggestive of the cause of hemospermia were obtained in the seminal vesiculogram and other clinical examinations. With daily injections of 20 cc. Strong Neo-Minophagen C, semen was not bloody on the semen examination carried out 1 week later. As the

bloody semen could not be demonstrated at all in the subsequent examinations, he was discharged from the hospital as being completely cured. However, in December of the same year, hemospermia recurred as a result of sexual excess, and he was admitted to the hospital again in January, 1962.

Status presens: The external genitalia were quite normal, as was also the prostate, and the seminal vesicles were not palpable or tender. The urine was clear.

Examination of the semen: Its color was chocolatebrown. The volume was about 3.0 cc. Sperm count was 65×10^6 /cc. Sperm motility was 50%. Red blood cell (+++). Pus cell (+). Culture showed negative tubercle bacillus and positive pathogenic staphylococcus. The semen was collected in two glasses, and the second glass was proved to contain more blood than the first.

Seminal vesiculogram: Seminal vesiculography using Endografin was carried out by vaso-instillation, but it revealed no noticeable anatomical anomalies nor inflammatory findings. The main duct was great in size,, and the diverticula developed normally. But in general, filling of opaque medium was not irregular and, therefore, the existence of inflammation was presumed (Fig. 5).

Diagnosis: Hemorrhagic spermatocystitis.

Operative findings: As hemospermia was so serious and various treatments failed seminal vesiculectomy was carried out. The seminal vesicles showed a slight adhesion with the surrounding tissues, and the content proved to be liquid of deep red in color.

Histological findings: The tunica in-

terna of the submucosal tissue was seen to proliferate and have edematous swelling with abnormal distribution of the arterioles and disorder of elastic fibers. The tunica media showed a slight degeneration and the tunica externa showed swelling-up of collagenous fibers and slight cellular infiltration. On the other hand, the mucosal folds partially showed hemorrhage together with epithelial changes (Fig. 6).

Remarkable proliferation of the connective tissue was seen in the mucosal folds, and swelling-up of the collagenous fibers was observed. There were no strong inflammatory changes observed, but the epithelium showed a fairly degree of degeneration. Besides, diffuse hemorrhage and intratubal bleeding were observed under the epithelium (Fig. 7).

He was perfectly well on the 22nd day after the operation. The sperm count was 2×10^6 /cc, and red blood cells completely disappeared at the time of discharge from the hospital.

Case 20. S. T., 24-year-old male, farmer.

Chief complaint: Hemospermia.

Past history: Gonorrhea and acute peritonitis.

Family history: Non-contributory. One child.

Present illness: He had been treated by a certain doctor under the diagnosis of cystitis since August 1961, but the discomfort in the posterior urethra and perineum did not disappear and finally he noticed hemospermia in the beginning of November of the same year, and came to the hospital.

Status presens: Serologic test for syphilis was negative. Tuberculin test was

positive. Bleeding and coagulation times were normal. The bladder, posterior urethra, testicles, epididymis, kidneys and ureters were all normal. Digital examination showed the normal prostate but above and to the right of the mid-line, a fairly large, soft, fluctuant mass could be felt. It was very tender.

Examination of semen: The semen was proved to be pink in color, including blood clots, about 2.0 cc in volume. The sperm count was $80 \times 10^6/\text{cc}$. Sperm motility 56%. Red blood cell (+). Pus cell (+). In the culture examination of the semen, tubercle bacillus was negative, pathogenic staphylococcus positive. The examination of the urine collected after prostatic massage revealed red blood cells, pus cells and non-motile sperm.

X-ray findings: The pyelogram, ureterogram, and urethrogram were all normal. Seminal vesiculography, carried out using 70% Endografin, demonstrated that the main duct was large in size and the diverticula were developed well on the left and the right seminal vesicle had great main duct but was a little poor in the development of the diverticula. In addition, there was pathological dilation of a little finger's tip size observed in the portion of the right ejaculatory duct. This was diagnosed as the pathological dilatation of the right ejaculatory duct, and the right seminal vesicle and cystic mass was surgically removed (Fig. 8).

The specimen revealed a cyst of a little finger's tip size, as demonstrated by x-ray examination, containing bloody mucous fluid.

Histological findings: The mucosal folds

were in disorder as a whole, and its epithelium showed pyknosis, degeneration and desquamation.

Besides, small bleeding was sporadically seen in the mucosal folds. Also in the lumen was diapedetic hemorrhage. However, there were a few inflammatory findings such as remarkable cellular infiltration in the submucosal and muscular layers (Fig. 9).

In the portion where the changes were the severest, most of the mucosal folds showed remarkable hemorrhage and fibrin precipitation, and the extensive hemorrhagic necrotic area were formed together with the infiltration of a small amount of neutrophils. The mild neutrophilic infiltration was seen also in the vicinity of such hemorrhagic and hemorrhagic-necrotic area (Fig. 10).

Examination of the semen carried out one month after the operation revealed no red blood cell, sperm count $40 \times 10^6/\text{cc}$ and sperm motility 80%. At the same time, the discomfort in the perineum was said to have completely disappeared, and the patient was discharged from hospital in cured condition.

Case 30. T. Y., 24-year-old male, company employee.

Chief complaint: Swelling of the left testicle accompanied by pain on pressure.

Past history & family history: Non-contributory. One child.

Present illness: The parotid glands had enlarged bilaterally several days before, and he had been under the treatment of acute parotitis. One day he had a contusion in the scrotum; since then he suffered from painful swelling in that portion, and finally came to the hospital. No abnormal findings

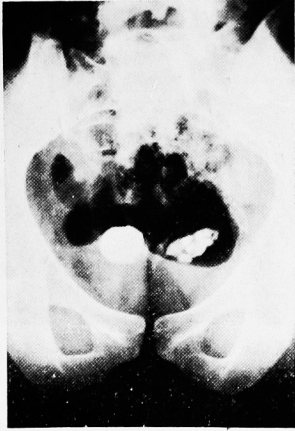


Fig. 1. Case 10. Seminal vesiculogram shows the pathological dilatation of the right seminal vesicle.

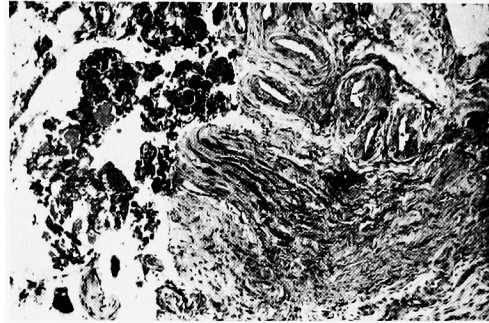


Fig. 4. Case 11. The histology of the ejaculatory duct. There was hemorrhage and slight necrosis of the mucosal epithelium and thickening of the wall of the arterioles. H & E, $\times 100$.



Fig. 2. Case 11. Seminal vesiculogram shows the pathological dilatation of the left ejaculatory duct with stones. De Graaf's phenomenon is negative.

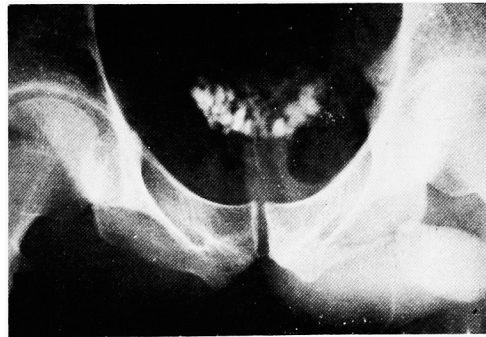


Fig. 5. Case 19. Seminal vesiculogram shows the presence of inflammation.

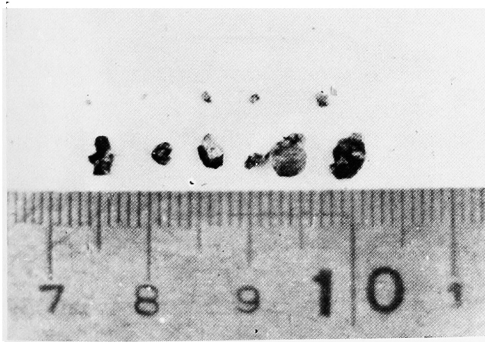


Fig. 3. Case 11. Gross specimen shows stones of the left ejaculatory duct.

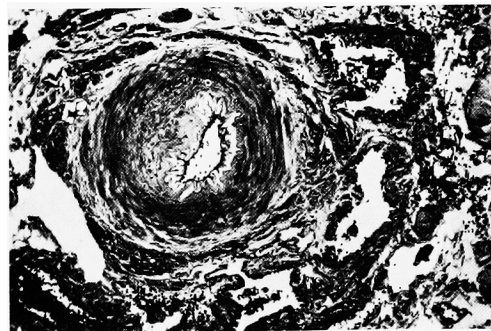


Fig. 6. Case 19. The histology of the left seminal vesicle. There was edematous swelling in the wall of the arterioles and the mucosal folds partially showed hemorrhage. H & E, $\times 100$.



Fig. 7. Case 19. The histology of the right seminal vesicle. Diffuse hemorrhage and intratubal bleeding were observed under the epithelium. H & E, $\times 100$.

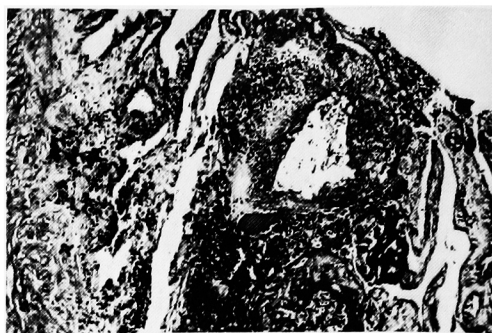


Fig. 10. Case 20. The histology of the left seminal vesicle. In the mucosal fold were observed the hemorrhagic necrotic lesions with infiltration of neutrophils. H & E, $\times 100$.

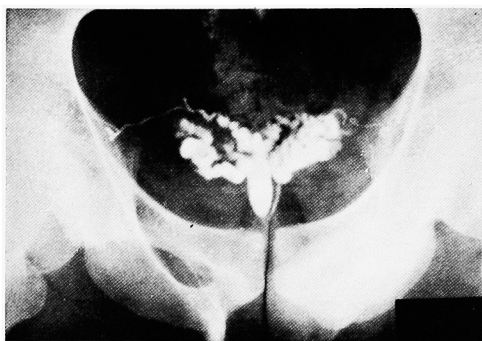


Fig. 8. Case 20. Seminal vesiculogram shows the pathological dilatation of the right ejaculatory duct.



Fig. 11. Case 29. Seminal vesiculogram shows dilatation of the ejaculatory duct with stones.

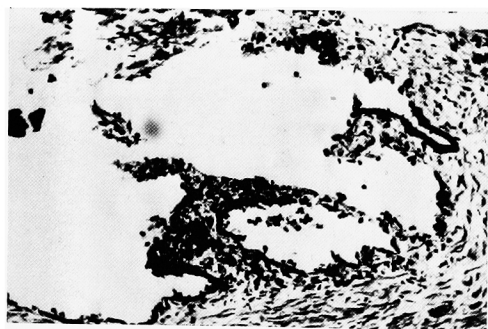


Fig. 9. Case 20. The histology of the right seminal vesicle. In the cavity was diapedetic hemorrhage and desquamation of the mucosal fold were observed. H & E, $\times 100$.

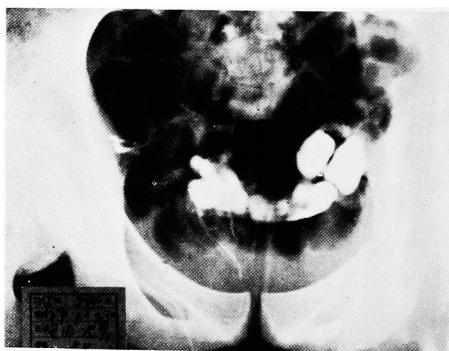


Fig. 12. Case 30. Seminal vesiculogram shows direct communication between the venous system and seminal vesicle on right side and asthenic dilatation complicated with chronic inflammation in both seminal vesicles.

in the chest and the abdomen. Upon rectal examination, the prostate was normal. Neither the seminal vesicle could be palpated. It was very tender.

The external genitalia were not remarkable except for a very tender left testicle. The left testicle enlarged to a size of a small egg.

Severe pain was complained on pressure, but no evidence of testicular rupture was obtained.

Laboratory reports : Urine examination showed no particular abnormality. Serologic reaction for syphilis was negative. Both bleeding and coagulation times were normal.

Examination of the semen showed about 4.0 cc in volume, pink in color, red blood cells (+), pus cells (++), sperm count 3×10^6 /cc, and sperm motility 0%.

On culture of the semen, tubercle bacillus was negative, and pathogenic staphylococcus positive.

The seminal vesiculogram revealed chronic infection with dilatation of both the seminal vesicles and very clearly showed that there was a direct communication between the venous system and the seminal vesicle lumen on the right (Fig. 12).

In this case, surgical operation was not carried out because of the patient's refusal. As a treatment, 1,200 mg erythromycin was daily administered for 5 days, and the swelling of the testicle subsided. Hemospermia also almost disappeared. Subsequent examination of the semen, however, demonstrated no improvement of sperm count and motility as compared with the first examination.

ARTICLE 2. ON TREATMENT OF HEMOSPERMIA

As hemospermia is of greatly manifold etiology and there are still many uncertain problems as to its details, no definite treatment has been established for this condition.

Bibliographically, a variety of methods have been reported for the treatment. They are summarized as follows :

I) Conservative treatment

1) massage of the seminal vesicles and prostate, 2) hormonal treatment (estrogen, androgen, adrenocortical hormones), 3) antibiotics, 4) hemostatics and 5) anti-allergic substances.

II) Surgical treatment

1) Seminal vesiculectomy, 2) extirpation of cyst or diverticulum, 3) removal of stones from the seminal vesicles, 4) drug infusion into the seminal vesicles by way of the vas.

As for the treatment using hormone preparations, Huggins & McDonald⁹⁾ reported in 1945 that estrogen was administered in 6 out of 20 cases with hemospermia and remarkable effect was obtained in 5 cases. Momose et al. reported a case of therapeutic success, and asserted that hormonal treatment was an interesting problem.

Many kinds of treatment have been tried against hemospermia of diverse etiology, but few methods have obtained so evident success in treatment as the surgical operation.

In author's cases hormone preparations, antibiotics, anti-allergic, hemostatics and surgical operations have been employed according to symptoms and causes ; for instance, fairly good results were obtained with administration of

antibiotics in the cases which showed positive results for pathogenic bacillus in the culture of the semen. Of the cases of unknown origin, 6 cases which were supposed to be attributable to allergy on the basis of their history and progress were rather satisfactorily cured by injection of 20 cc Strong Neo-Minophagen C and administration of adrenocortical hormone, but some cases had recurrence of the condition after once cured and were compelled to undergo surgical treatment. The administration

of anti-allergic substances will be considered to be one of the reliable method in the future. Symptomatic therapy using hemostatics has been generally employed in many patients.

It was employed in 13 cases, and 7 cases of them were almost cured. Its cure rate was as low as 54%. It seemed that hemostatics could not be greatly expected in most cases.

The therapeutic methods employed in my cases and their effects are illustrated in Table 4.

Table 4. Method and effect of treatment.

Method of treatment		Patient	Cure	Per cent
Conservative treatment	Hemostatics	13	7	53.9
	Anti-allergic substances			
	Strong Neo-Minophagen C	5	4	80.0
	Adrenocortical hormone	1	1	100
	Antibiotics	7	5	71.4
	Estrogen	1	1	100
Surgical treatment	Seminal vesiculectomy	4	4	100
	Excision of pathologically dilated portion and lithotomy	1	1	100
	Vaso-instillation of antibiotics	12	8	66.7

CHAPTER 4. RESULTS OF EXPERIMENTS

ARTICLE 1. ON SPERM MOTILITY

As sperm motility was observed to lower in many of my cases with hemospermia, the author carried out the following experiments in order to investigate the relationship between homospermia and sperm motility.

1) Methods

1) Materials

A) Human semen

a) The semen of a healthy man was collected in a sterile manner by a manual

method after 4 days' abstinence. The semen in this experiment was required to have sperm count of more than $80 \times 10^6/\text{cc}$ and motility of more than 80%.

b) The semen was collected from the patients suffering from spermatocystitis or prostatitis as well as hemospermia by a manual method.

Irrigation of the urethra was carried out prior to the collection of the semen so as to avoid the possible invasion of bacilli from other parts.

B) Human blood serum

The blood of the healthy men (the

same persons whose the semen had been gathered) was collected, regardless of blood groups, from the cubital vein. The serum was isolated by centrifugation.

2) Method of experiment

Experiment 1: After the collected semen of a healthy man was liquefied, 0.5 cc of the semen and 0.5 cc of healthy serum were poured into 5 test tubes. As a control, mixture of 0.5 cc of the semen and 0.5 cc of the seminal plasma (obtained by the centrifugation of the healthy semen) was employed. These tubes were left at room temperature, and then the sperm motility was examined microscopically 1, 2, 3, 4 and 6 hours later.

Experiment 2: 0.5 cc of the seminal plasma obtained by centrifugation of the semen collected from the patients with spermatocystitis and prostatitis was added to 0.5 cc of the semen of a healthy man, and then left at room temperature. The changes in the sperm motility was examined 1, 2, 3, 4 and 6 hours later.

II) Results of the experiments

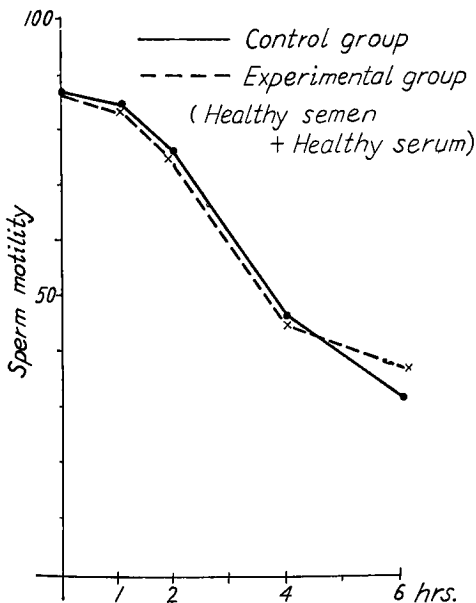


Fig. 13. Effect of serum on sperm motility.

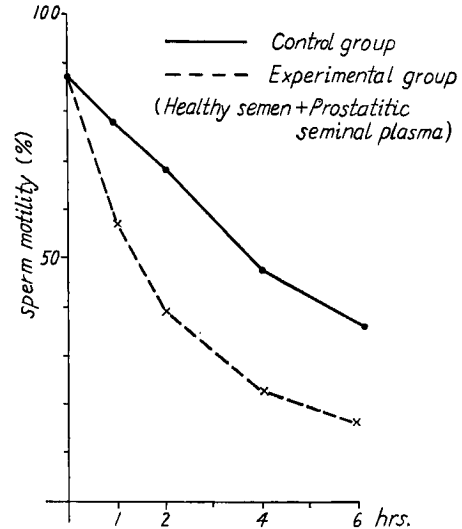


Fig. 14. Effect of seminal plasma of prostatitis on sperm motility.

In the Experiment 1, as seen in Fig. 13, the influence of the serum upon the sperm motility was very mild and preserved its activity, and there was little difference from the control in the curve of the mean motility rate.

In Experiment 2, as seen in Fig. 14, the curve of the mean motility rate showed evident changes. It was remarkably demonstrated that the seminal plasma of the patients with spermatocystitis and prostatitis had action of inhibiting the motility of sperm.

ARTICLE 2. ON SEMINAL VESICLE ALLERGY

A phenomenon of allergy in the various organs has been much investigated by many doctors^{(8) (9) (50) (51) (52) (53) (54) (55) (56)}. As for the allergic changes in the seminal vesicles, this organ has long been said to be the tonsil of the genital organs and regarded as the primary lesion of some systemic allergic diseases. It has fairly great significance. It is a well known fact that the mucosa of the

seminal vesicles has histologically same properties of the absorbing organs. Ishigami (1955, 58)^{57) 68)} experimentally demonstrated that absorption of bacilli in the seminal vesicles was very remarkable as compared with the other viscera and that the bacillary invasion into the blood stream was made in rather early stages. He also reported that the bacillus-absorbing power of the seminal vesicles was by far weaker in immunized guinea pigs than in untreated ones.

It is commonly known that intracutaneous injection of the same antigen generally leads to the so-called Arthus' phenomenon in the site of the immunized animal, being helpful to the prevention of the antigen transition into the blood and further to the prevention of systemic anaphylaxis.

From these facts and clinical experiences, it is suggested that there are some relations between hemospermia and allergy and it seems to be of great significance to observe these problems.

Hereupon, author experimentally induced anaphylaxis, both Schwartzman and reversed anaphylactic reactions, in the tissue of the seminal vesicles in the rabbits, and examined the resulting histological changes.

I) Schwartzman phenomenon

1) Experimental animals: Healthy adult male rabbits of about 2 kg body weight were used (bred on the same food for 1 week).

2) Materials: *Escherichia coli* communior was cultured at 37°C for 24 hours on an ordinary slanted agar medium (pH 7.4). The bacterial body was washed and made to float in the physiological saline solution in the ratio of 10 mg/ml.,

and was further cultured in an incubator at 37°C for 24 hours, and then absorbed and filtrated by a sterilization filter. The resulting filtrate was kept in a refrigerator to use as Schwartzman's filtrate.

3) Methods

As a preliminary injection, 0.25 cc Schwartzman's filtrate was injected into the seminal vesicles by way of the vas (Group 1). In Group 2, the seminal vesicles were surgically exposed and then 0.2 cc of the Schwartzman's filtrate was injected into the submucosal part of the seminal vesicles. At the time of operation, disinfection and other necessary procedures were strictly performed.

Twenty-four hours after the preliminary injection, the provocative injection of the Schwartzman's filtrate was made into the marginal ear vein of the rabbits in dosage of 1 cc per kg body weight. The animals were sacrificed 24 hours after the provocative injection. The seminal vesicles were removed and fixed with 10% formalin, and then processed for a paraffin section. Hematoxylin-eosin staining was made for microscopic examination.

In addition, as a control, the seminal vesicles of the rabbits were sacrificed 24 hours after the preparatory injection were routinely fixed and stained.

4) Results

Histological findings: In the histological observation, remarkable changes were found as follows.

Group 1) The mucosal folds showed slight atrophy, but not significant changes. An conspicuous hemorrhage was diffusely observed in a part of the submucosal layer. Infiltration of some

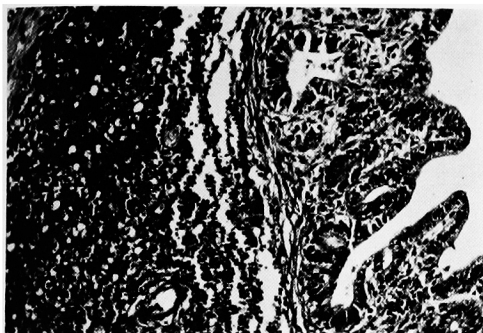


Fig. 15. The histological findings of the rabbit seminal vesicle in Schwartzman phenomenon (Group 1). The submucosal layer showed diffuse hemorrhage and infiltration of pseudo-eosinophilic leucocytes. H & E, $\times 200$.



Fig. 18. The histological findings of Schwartzman Ph. (Group 2). The submucosal layer was remarkably sanguineous due to extensive hemorrhage. H & E, $\times 100$.

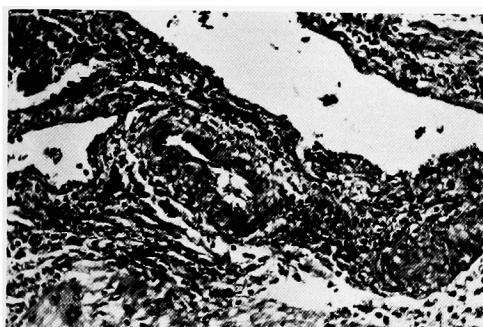


Fig. 16. The histological changes of Schwartzman Ph. (Group 1). Intense edema of subendothelial area in the arterioles and remarkable infiltration of pseudo-eosinophilic leucocytes in its surrounding were observed. H & E, $\times 200$.

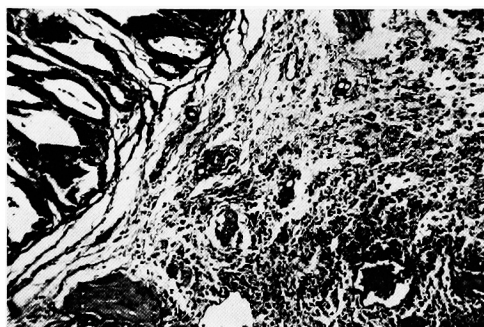


Fig. 19. The histological findings of Schwartzman Ph. (Group 2). In the submucosal layer, edema and infiltration of the pseudo-eosinophilic leucocytes were demonstrated. H & E, $\times 100$.



Fig. 17. The histological findings of Schwartzman Ph. (Group 1). Hemorrhagic necrosis accompanied by fibrin deposit and hemorrhagic lesion was observed in the mucosal layer. H & E, $\times 200$.

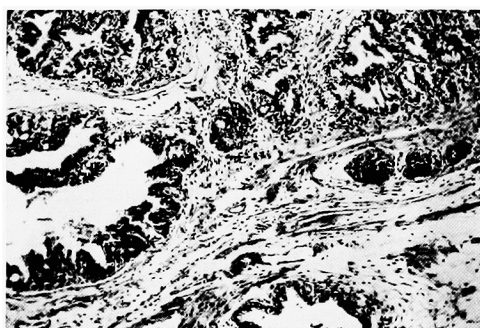


Fig. 20. The histological findings of control group in Schwartzman Ph. (Group 1). No noticeable changes. H & E, $\times 100$.

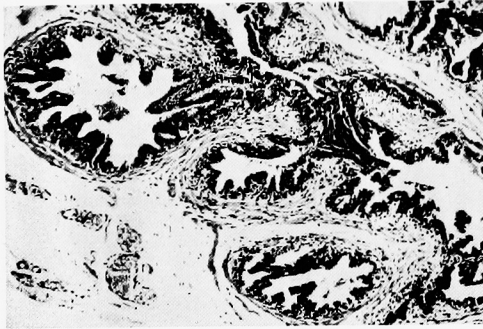


Fig. 21. The histological findings of control group in Schwartzman Ph. (Group 2). No significant changes. H & E, $\times 100$.

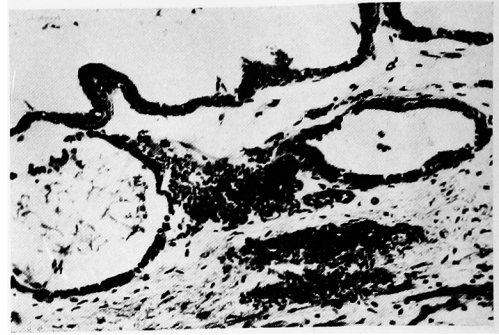


Fig. 24. The histological findings of reversed anaphylaxis (Group 2). Localized hemorrhagic lesion and infiltration of pseudo-eosinophilic leucocytes were observed in the submucosal layer. H & E, $\times 100$.

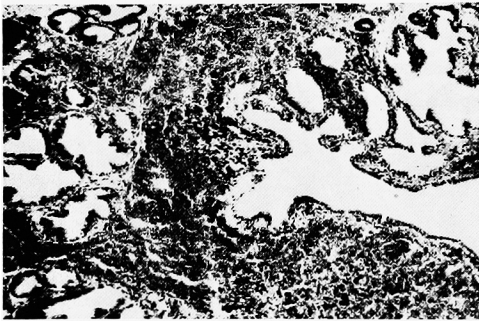


Fig. 22. The histological findings of the rabbit seminal vesicle in reversed anaphylaxis (Group 1). The stroma was remarkably sanguineous due to extensive hemorrhage. H & E, $\times 100$.

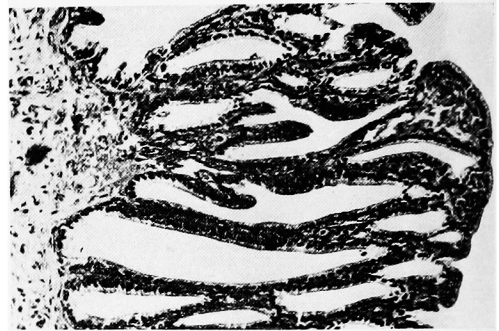


Fig. 25. The histological findings of reversed anaphylaxis (Group 2). The mucosal folds were observed to be remarkably hemorrhagic. H & E, $\times 100$.

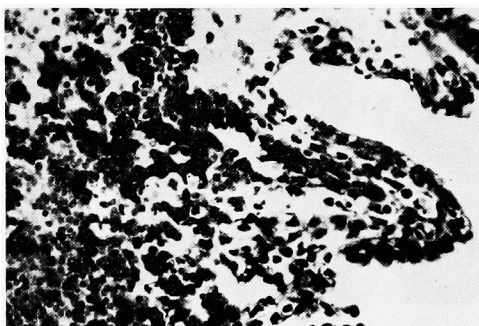


Fig. 23. High magnification of Fig. 22. There was observed desquamation of the mucosal fold and remarkable hemorrhage in the stroma. H & E, $\times 400$.



Fig. 26. The histological findings of control group in reversed anaphylaxis (Group 1). No significant change. H & E, $\times 100$.

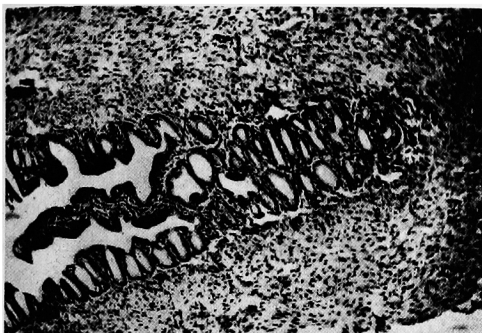


Fig. 27. The histological findings of control group in reversed anaphylaxis (Group 2). No significant change. H & E, $\times 100$.

pseudo-eosinophilic leucocytes was observed in the hemorrhagic lesion (Fig. 15). The arterioles of the submucosal layer showed intense edema in the subendothelial area, edema and roughness in the wall, with remarkable infiltration of the pseudo-eosinophilic leucocytes in its surrounding area (Fig. 16). In another part of the mucosal folds, not only considerable edema and diffuse diapedesis of low grade were observed, but also hemorrhagic necrosis accompanied by fibrin precipitation and hemorrhagic lesions appeared in parts. Infiltration of the pseudo-eosinophilic leucocytes was not particularly observed in the lesions of hemorrhage and hemorrhagic necrosis, but slight reactions of the pseudo-eosinophilic leucocytes were observed in their surroundings.

Group 2) The mucosal folds and submucosal layer showed so extensive hemorrhage and hemorrhagic necrosis that the lumina of the folds looked as if they had been isolated in a bloody area. Such bleeding was, however, not seen in the muscular layer, with few reactions of the pseudo-eosinophilic leucocytes in its surrounding (Fig. 18).

In the submucosal layer, edema and

pseudo-eosinophilic infiltration were demonstrated to be wide spread, together with thickening of the wall of the arterioles and slight bleeding. The submucosal layer showed a considerable thickening due to these changes (Fig. 19).

Histological findings of the control group :

Group 1) There were no noticeable changes observed in the mucosal folds and the submucosal layer, except for slight edema and slight infiltration of small round cells (Fig. 20).

Group 2) Just as in Group 1, both edema and cellular infiltration were mild, and no significant changes were obtained (Fig. 21).

II) *Reverse anaphylaxis*

1) Experimental animals :

Guinea pigs; 30 healthy adult male guinea pigs of about 250 g weight were used (bred on the same food for 1 week).

Rabbits; Healthy adult male rabbits of about 2 kg weight were used (bred on the same food for 1 week).

2) Materials ; The heart-puncture blood, added with 2.8% sodium citrate, was centrifugated for 10 minutes at the rate of 3,000 rounds per minute. All the supernatant fluid and white blood cells were eliminated from the deposit. Centrifugation was carried out again with sterilized physiological saline solution added. Such procedures were repeated 5 to 6 times until the resultant supernatant fluid became transparent. The red blood cells contained in the deposit were prepared into a 5% physiological saline suspension. 5 cc of this 5% suspension were injected eight times into

the abdominal cavity of the guinea pigs every other day. Total blood was collected 1 week after the last immunization, and serum was isolated from the collected blood. The serum was treated by heat immobilization for 30 minutes at 56°C, and the titre of hemolysin to rabbit erythrocyte was determined. In other words, 0.25 cc of the anti-serum was added to one of the test tube filled with 0.25 cc physiological saline solution, and then multiple dilution was repeated in tubes in this way. In the next place, complement (10-fold diluted serum of normal guinea pigs) and 2.5% rabbit serum 0.25 cc each, were put into every test tubes and then heated in the water bath of 37°C. The determination was performed 1 hour later. The maximum dilution causing complete hemolysis was regarded as the hemolysin value of the relevant anti-serum. In 1:64 diluted serum, hemolysin was read as positive.

3) Method:

Group 1) 0.2 cc of the guinea pig serum immunized with rabbit red cell was injected into the seminal vesicles by way of the vasa after laparotomy on the rabbits. Group 2) In the other group, the injection was made directly into the submucosal layer of the seminal vesicles under surgical exposure.

Disinfection and other necessary procedures were strictly performed. The animals were sacrificed 24 hours after the injection to examine the resultant histological changes in the seminal vesicles. For such purpose, the extracted specimens were fixed with 10% formalin and processed for paraffin sections. Hematoxylin-eosin staining was made for the microscopic examination.

Besides, serum of normal guinea pigs was used as a control in the same manner as in the experimental groups. The animals were also sacrificed to obtain the specimens.

4) Results: In the histological observation, the following remarkable changes were found.

Group 1) The stroma of the seminal vesicles was remarkably sanguineous due to severe hemorrhage. Slight reaction of pseudo-eosinophilic leucocytes was observed in its surrounding (Fig. 22). In the submucosal layer, small round cells were seen to infiltrate mildly but diffusely associated with bleeding and edema (Fig. 23).

Group 2) Localized hemorrhagic lesions and infiltration of pseudo-eosinophilic leucocytes were observed in the submucosal layer. A moderate edema was observed, but no remarkable inflammation was produced (Fig. 24). In the mucosal folds was observed very remarkable hemorrhage, and in the submucosal layer a significant edema (Fig. 25).

Histological findings of the control group:

Group 1) No significant changes were obtained except for slight edema in the submucosal layer (Fig. 26).

Group 2) No significant changes were detected (Fig. 27).

CHAPTER 5. DISCUSSION AND SUMMARY

The literatures concerning hemospermia and the outlines of clinical symptoms and the results of various examinations in 33 cases of hemospermia have been presented. In addition, the results of fundamental experiments on the seminal

vesicle allergy were also reported in order to use as a mean of elucidating complicated clinical pictures of this condition. Many of the clinical cases were devoid of pain and other concomitant symptoms besides hemospermia. Five cases, complaining of infertility, did not notice of this condition before they were pointed out to have it on the semen examination. Hemospermia gives a fairly large psychological burden on all the patients; diminished sexual libido has been evidently complained of in many cases.

Momose et al. pointed out this fact, and stated that it was uncertain whether this condition might be attributable to organic disturbances or caused by psychoneurotic factors. In author's experience, the latter, i.e. the psychoneurotic factors should be emphasized. Ochiai (1960)⁵⁹⁾ stressed the disturbance of sexual function as one of the clinical symptoms of chronic spermato cystitis and prostatitis, saying that hyposexuality and hemospermia were these symptoms with occasional association of depression and signs of neurosis which were often troublesome.

Occupations of the patients are fairly widely ranged, but many of them are company employees, especially the persons who engage in desk works. It is, however, difficult to think that the overburden of the patient's job might be the cause of such condition. As to the personal history, pulmonary tuberculosis and gonorrhoea are found in nearly half of the cases, but it is not clear whether or not such past history might be a direct cause or predisposing to this condition, for we have not experienced the

cases suggestive of genital tuberculosis particularly of seminal vesicular tuberculosis. Nevertheless, the relation between genital tuberculosis, especially tuberculosis of the seminal vesicles, and hemospermia has so far been fairly emphasized; for instance, Eisendrath & Rolnick, Hugh Young, Parker, Voelcker and others stated that tuberculosis was one of the important factors in the occurrence of this condition. In recent years, seminal vesiculography which was devised by Belfield became widely available; and the diseases of the seminal vesicles have consequently become able to be diagnosed more easily and correctly. Even in a normal seminal vesiculogram, however, the seminal vesicles assume various complicated aspects and not always show symmetry in the same person. It is therefore necessary to be deliberate in making diagnosis, as commonly admitted. Mukoyama (1954)⁶⁰⁾ observed the changes of the seminal vesiculograms in 42 out of 58 cases with genital tuberculosis, and demonstrated the diagnostic value of the seminal vesiculography. In author's experience, there was not a single case which showed a tuberculous change on a seminal vesiculogram, but 9 cases were radiographically seen to have pathological dilatation of the seminal vesicles and the terminal portion of the vas. Among these cases, the stone shadow was observed in 2, communication between the vein and the seminal vesicle in 1 and inflammatory changes in 2 case.

Such anatomical abnormalities are supposed to be the cause of hemospermia as we have already discussed above. Kusunoki performed seminal vesiclec-

tomy in the patient with diverticula of the seminal vesicles accompanied by hemospermia, and Ochiai carried out seminal vesiculectomy in a patient with hemospermia accompanied by cyst of the seminal vesicles. Both stated that the histological finding was almost normal and that hemospermia disappeared clinically. In other literatures, the case of pathological dilatation were reported to be cured by seminal vesiculectomy. In author's cases 3 were successfully cured by operation.

As seen from the above discussion, a close relation between the pathological dilatation of the seminal vesicles and the terminal vas and hemospermia can not be denied. In the case of pathological dilatation of the seminal vesicles and the terminal vas, this anatomical abnormality give rise to the retention or stagnation of the semen facilitating secondary infection and to distention of the cystic wall producing a state favorable to hemorrhage. Such is thought to be the cause of the occurrence of hemospermia. As for the treatment of the cystic dilatation, there are some reports on the cases which had been cured by the puncture from the rectum, but its radical treatment is still the extirpation of the seminal vesicles including the dilated portion. On this occasion, as Ishigami (1960)^{61) 33)} has already pointed out, it is advised to cure the patient with his fertility preserved by removing only the dilated portion if the dilated portion is existing as a lateral branch to the transportation route of the sperm. In one of the cases, calculi were simultaneously removed with the excision of the dilated portion of the ejaculatory

duct, resulting not only in eliminating hemospermia but also in obtaining improvement of sperm findings and further the birth of a healthy boy.

It is very interesting that Bauer (1956)⁶²⁾ reported a case of this condition complicated with infertility in which fertilization was successfully done after the extirpation had been performed. The extirpation has much to be greatly expected as a treatment for infertility.

An interesting fact is that all the married patients, including 5 cases of infertility, have not caused pregnancy since this condition was detected. As there were many cases which were considered to be unable to fertilize from the seminal findings, such dilatation might be attributable to hypofunction of the accessory organs due to infection, and to the lowered motility of sperm due to intracystic stagnation. According to Momose's report, all his 12 married patients failed to establish pregnancy since the detection of this condition; then, the relation between hemospermia and infertility was suspected. Finger⁶³⁾ stated that the sperm was not seen at all in the semen or, if any, became non-motile in the case of hemospermia. Besides, Peyer⁶⁴⁾ and Casper⁶⁵⁾ reported that hemospermia could be the cause of male infertility. I am in favor of this opinion on the basis of own experiences.

The experiments were also performed about the influence of healthy human serum, and the seminal plasma obtained from the patients with seminal vesiculitis and prostatitis upon the motility of the sperm.

The serum itself did not exert any great influence on the sperm motility

and preserved its activity, but the seminal plasma obtained from the patients with seminal vesiculitis and prostatitis evidently suppressed the sperm motility. With respect to this problem, Kurzrok (1953)⁶⁶⁾ demonstrated the promoting effect of the liquor folliculi on the sperm motility. Jöel & Kornhanser (1956)⁶⁷⁾ recognized that the antibiotics had favorable effects on the preservation of sperm activity. Sanada (1959)⁶⁸⁾ demonstrated the promoting effect of the serum on the sperm motility, but Momose could not obtain any change when he examined the effect of blood on the sperm motility. Sakakura (1961)⁶⁹⁾ repoted the promoting effect of the various antibiotics and protein on the sperm motility. Moriwaki³⁶⁾ did not recognize any active sperm but found oligozoospermia in the patient with hemospermia who had already 2 children. To be of interest, he interpreted that the inflammation of the seminal vesicles was accelerated by the added postnatal factors. Author considered that the morbid abnormality of the secretions from the accessory organs due to secondary infection might exert an unfavorable influence on the sperm motility rather than the serum itself.

Takeda (1959)⁷⁰⁾ reported that the fructose content in the secretions from the accessory sex organs was great when the functions of those organs were active, and small in the case of hypofunction; and he concluded that the fructose amount showed alteration in accord with the organic changes of the accessory sex organs. The close relationship between the fructose amount of the semen and the sperm motility is

clearly demonstrated at present. Therefore, it would be possible from the above informations to elucidate the relation between hemospermia and fructose amount and further the relation between bloody semen and the sperm motility. The fructose amount of the semen collected from the patients with seminal vesiculitis and prostatitis was both within the range of 130 mg/dl to 200 mg/dl in my experiments. There are considerable differences in the normal value of the fructose content according to the investigations individually.

Yamamoto (1962)⁷¹⁾, my colleague, regards the normal mean value as 350 mg/dl. When this value is compared with the fructose value of the semen of the spermatocystitis and prostatitis in the present experiments, the latter is seen to be lowered to a fairly large extent.

Lowered motility of the sperm in the patient with hemospermia can be explained from this point of view. Mitsuya (1954)⁷²⁾ reported that the fructose content of the semen was 170 mg/dl in the patient with hemospermia. Nishimura (1953)⁷³⁾ determined the fructose content of the semen in 2 cases of tuberculosis of the seminal vesicles and of the prostate, and reported that it lowered to 105 mg/dl and 12 mg/dl, respectively. In author's experience improvement of seminal findings was observed in many cases after the treatment of hemospermia, and the sperm motility was elevated in the cases with necrozoospermia or with lowered sperm motility. The increase in the sperm count was, however, not observed in the cases with azoospermia and oligozoospermia. Ishigami et al.

(1962)⁷⁴⁾ examined the semen collected from 9 patients with prostatitis, and reported that no great changes were obtained in the volume of the semen and its fructose content, but that the sperm motility was considerably lowered as compared with the normal one. He further stated that such changes were in accord with the degree of diseases and process as well as the individual factors, and that the motility was improved together with the improvement of underlying diseases. It is well known fact that the seminal vesicles have great significance as a primary lesion of various systemic diseases and further that it is even said as the tonsil of genital organs. Ishigami^{75) 76) 77)} made a preliminary examination on the absorption of various foreign proteins in consideration of the immunological significance of the seminal vesicles, and then investigated the absorption of various antibiotic substances, bacteria and endocrine secretions from the clinical and endocrinological points of view. Ishigami⁷⁸⁾ also reported that the injection of a sulfamine solution directly into the seminal vesicle caused severe skin eruptions than its hypodermal injection in the patient with fixed sulfamine eruption.

Being interested in such reports and in an attempt at elucidation of the relationship to allergy as one of the causes of hemospermia, anaphylaxis of the seminal vesicles, both Shwartzman phenomenon and reversed anaphylaxis, were studied, and remarkable hemorrhage, hemorrhagic necrosis, cellular infiltration and further degenerative changes of the stroma and blood vessels were

recognized in the histological findings on the seminal vesicles, which were all considered to be ascribed to allergic mechanism. These histological changes have many similarity with the findings of seminal vesicles surgically removed from the patients with hemospermia. Clinically, the anti-allergic agents were employed in 6 cases with complete cure in 5 cases. Author noted some allergic infections in the history, physical examination and progress of those patients.

When the therapeutic results are taken into consideration, relation with allergy can be conceived. In this way, the author could demonstrate based on the clinical findings and fundamental experiments that allergy in the seminal vesicles can be one of the cause of hemospermia, but we would like to expect further investigations in future.

Besides, it can not be denied that the various hemorrhagic dispositions can be one of the causes of hemospermia, although none of my patients showed prolonged bleeding and coagulation times. Classification of the etiological factors in diseases is very important. Finally, based on the etiological studies of other investigators in the available literatures and my own experiences, the following classification of the causes of hemospermia was established.

I. Hemospermia due to organic disturbances (Seminal fract)

- 1) Anatomical and morphological abnormalities
 - a) Pathological dilatation of the seminal vesicles and the vas deferens
 - b) Direct communication between the venous system and the seminal

vesicle

- 2) Non-specific inflammation
 - a) Bacterial inflammation
 - b) Aseptic inflammation
- 3) Specific inflammation
 - a) Tuberculous inflammation
 - b) Syphilitic inflammation
- 4) Stones
- 5) Tumors
- 6) Traumas

II. Hemospermia due to functional disturbances

- 1) Allergy
- 2) Sexual neurasthenia
- 3) Sexual excess or gross masturbation
- 4) Hemorrhagic dispositions

As the etiology of so-called essential renal bleeding has been discussed in various ways, there are many cases of hemospermia of which the cause can not be elucidated even by clinical examinations.

Such hemospermia as provides with no clue for diagnosis might be called so-called essential hemospermia. The cause of such hemospermia may be explained by the organic diseases but seems to be closely associated with the autonomic nerve dysfunction and further with the problem of allergy. Further examinations are highly called for in future on this problem.

CHAPTER 6. CONCLUSION

On the 33 cases of hemospermia, clinical and statistical observations were reported, and the patho-histological investigations were carried out in 3 cases. Besides, some fundamental experiments were performed. The conclusions are as follows :

(1) Hemospermia was mostly observed among the persons of the third and fourth decade reaching 75% and the average age was 34.

(2) Among the patients with hemospermia, 36.3% evidently showed azospermia, oligozoospermia and necrozoospermia. Lowered sperm motility was also observed in most of the other cases.

(3) No pregnancy had been established in all the cases since the occurrence of hemospermia.

(4) The effect of healthy human serum and the seminal plasma of the patients with spermatoecystitis and prostatitis (inclusive of hemospermia) on the motility of healthy sperm was examined. The serum had preserved sperm motility, but the seminal fluid of spermatoecystitis remarkably suppressed motility of normal sperms.

(5) Shwartzman phenomenon and reversed anaphylaxis were experimentally provoked in the rabbit seminal vesicles, and allergic changes could be demonstrated in the seminal vesicles.

These histological changes have many similarity with the findings of seminal vesicles removed from the patients with hemospermia.

Based on these findings, the author could demonstrate that allergy in the seminal vesicles can be one of the causes of hemospermia.

(6) It is thought that the pathological dilatation of the seminal vesicles and the end of the vas deferens can be one of the factors of hemospermia, and the significance of seminal vesiculography as a routine examination for hemospermia was confirmed.

(7) Based on the bibliographic consid-

rations and own clinical findings, the classification of the causes of hemospermia are advocated.

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血 精 液 症 に 関 す る 研 究

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血精液症は諸種精路系疾患の主症状或は随伴症状として見られる一症候である。然しその自覚症状が差程顕著でない事から等閑にふされていることが多く、その病因及び治療については尚未解決の点が少くない。

著者は本学泌尿器科を訪れた患者の内、本症を認めた者に対し、その臨床的統計的観察を行うと共に、それに関連した2～3の基礎実験を試みたので報告する。

I 臨 床 的 観 察

過去4カ年間における外来患者中血精液症は33名(0.84%)に認め、平均年齢は34才、精神労働者に多い。既往歴は肺結核、淋疾が多いが本症との直接的関係は明らかでない。精液細菌培養検査において病原性葡萄球菌を多数に認めた。尚全例に精囊腺X線撮影を行い、9例に精

囊腺並びに精管末端部の異常拡張像を、又2例にこれに合併した結石症例、及び血管との交通例を経験した。更にこれらの手術的所見及び病理組織学的検索より、これらの解剖学的異常が血精液症の直接的原因となつている事が判明した。又当該拡張部の剔出術により血精液症の消失は勿論、健常児の出産を見た1例を経験している。以上の如き器質的障害によるものの他、その原因不明のものに対し抗アレルギー剤を投与して著効を収める症例のある事を認めた。この事実より著者は本症発生機序の一因として、アレルギー性因子の関与することを推測し、下記の如き基礎実験を行つた。治療には病因によつて抗生物質（全身及び局所投与）、抗アレルギー剤、止血剤、エストロゲン投与及び手術的療法等を行い、その各々の効果について検索を行つた。

Ⅱ 基礎実験

1. 人血清及び精囊腺 前立腺炎患者精漿の精子運動に対する影響について

症例の既婚者全例が本症発現後妊娠成立を見ていないこと、及び精液所見において精子数、精子運動性から明らかに妊孕不能と考えられる症例が多い事実から精子運動性に及ぼす精液中の諸因子を追求する目的で次の如き実験を行った。

(実験方法)；第1群には健康男子精液に本人血清を1：1に混和し、第2群には精囊腺前立腺炎患者精漿を同様混和し、各々時間的推移による運動性の変動を対照群と比較検討した。

(実験成績)；第1群では精子の運動性に何等阻害作用を認めなかつたが、第2群では明らかに阻害作用を認めた。これによつて本症に於ける精子の運動性低下は血液の混和によるよりも、むしろ精路の器質的变化とこれに伴う副性器分泌物の病的変化が影響するものと推察される。

2. 精囊腺アレルギーについて

実験的に家兎精囊腺にアナフィラキシーを惹起せしめ、その病理組織学的検索を行うと共に、臨床的に得られた本症患者の精囊腺組織像と比較検討した。

(実験方法)；a) シュワルツマン現象、体重2kg前後の雄性成熟家兎を用い、第1群には大腸菌培養液(Sh. 汚液)0.25ccを経精管的に精囊腺内に注入し、第2群には精囊腺粘膜下に注射した。この準備注射後24時間目に両群

共耳静脈より1kg当り1.0ccのSh. 汚液を注射し、24時間後の精囊腺を剔出、ヘマトキシリン・エオジンによる組織学的検索を行った。

b) 逆アナフィラキシー。a群同様の家兎を用い、抗血清として抗家兎赤血球モルモット血清を使用した。第1群には抗血清0.25ccを経精管的に精囊腺内に注入し、第2群には同粘膜下に注射、24時間後、精囊腺を剔出しa実験群同様組織学的に検索した。

(実験成績)；Sh. 現象組織像と逆アナフィラキシー時の組織像は精囊腺において同様の所見を呈し、粘膜層及び粘膜下層に出血、出血壊死、浮腫、細胞浸潤及び血管壁の変化が見られた。これらの所見は臨床的に剔出した本症精囊腺組織像と全く類似している。

Ⅲ 結 語

1) 血精液症33例を経験し、臨床的統計的観察及び文献的考察を行い、その病因、治療等について述べ、又それに附随した2～3の基礎的実験を行った。

2) 精囊腺X線像から、精囊腺及び精管末端部の異常拡張が本症の成因となる事、及び精囊腺アレルギーが重要な病因の一つである事を認めた。

3) 血精液症患者の精子運動性障害の因子として炎症性副性器分泌物が働くことを実験的に立証した。

4) 家兎を用いての実験的精囊腺アレルギー反応によつて、臨床剔出精囊腺組織像と類似した所見を得たことから血精液症の一病因として精囊腺アレルギーのあることを認め得た。